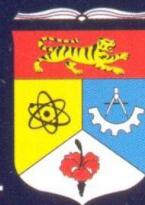


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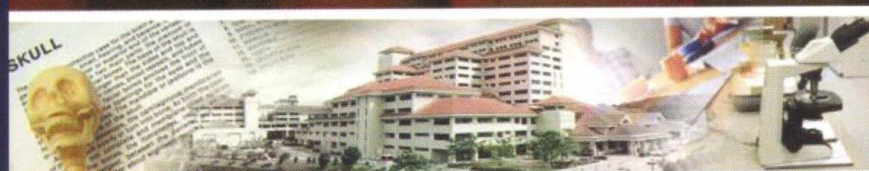
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INVOLVEMENT OF p16^{INK4a} IN SENESCENT FIBROBLAST MORPHOGENESIS

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Background:

During cellular senescence, normal human diploid fibroblasts (HDFs) change their morphology from spindle shape to enlarged, flattened and irregular shape. Besides, senescent HDFs express elevated levels of p16^{INK4a} which is known as cyclin-dependent kinase inhibitor (CDKI). The aim of this study was to determine the involvement of p16^{INK4a} in HDFs senescent morphogenesis.

Materials and Methods:

Serial passaging was done and the number of population doublings (PDs) was monitored until HDFs reached senescence (passage 30). Senescent cells were transfected with p16^{INK4a} siRNA for 48 hour and subsequently the RNA was extracted for real time RT PCR analysis.

Results:

Our results showed normal human diploid fibroblasts enter senescence state after 50-60 population doublings. Senescent HDFs showed morphological changes with the presence of senescence-associated β -galactosidase and increased p16^{INK4a} expression. However, senescent HDFs transfected with p16^{INK4a} siRNA showed downregulation of p16^{INK4a} and changes of morphology from senescent morphology to morphology of young cells with the presence of small and spindle shaped fibroblasts.

Conclusion:

In conclusion, increased of p16^{INK4a} expression is correlated with senescent morphology. However, inhibition of p16^{INK4a} expression may delay the onset of cellular senescence.

Keywords:

siRNA, cellular senescence, p16^{INK4a}